Introduction

Unlike most organs, the skin is in constant contact with the external environment. The skin ensures the body's integrity by preserving internal fluids and electrolytes, maintaining thermoregulation, and protecting against physical injury and entry of harmful agents. Because the skin has such a prominent and protective role, many factors affect it adversely, including mechanical agents (friction, vibration, pressure, and trauma); physical agents (heat, cold, and radiation); biologic agents (plants, insects, animals, and microbes); and a variety of chemical agents.

The large number of chemicals in the home and workplace and the accidental and intentional releases to air, water, and soil potentially allow ever-increasing contact with chemicals in the environment. Dermatitis from chemical exposures in the workplace accounts for about 30% of all reported occupational illness; the prevalence of skin lesions due to chemicals encountered outside the workplace (i.e., environmental exposures) may never be known.

Seven common skin conditions that can have environmental etiologies are presented in this monograph. Accurate diagnoses and identification of etiologies are necessary, not only to properly treat skin diseases, but also to prevent future occurrence of disease or exposure.

Familiarity with the vocabulary of dermatology is helpful in understanding this specialized topic. A glossary of terms begins on page 42.

(a) What are the most likely nonoccupational etiologies for four of the more common skin conditions: irritant and allergic contact dermatitis, urticaria, and photosensitivity?

(b) What are the most effective treatments and preventive measures for each of these skin conditions?

Answers begin on page 39.

Case 1 — Irritant Contact Dermatitis

A husband and wife consult you because of skin rashes that have developed since they began renovating a recently purchased older home. They have no history of skin problems.

The man complains of severe itching of the hands and an erythematous rash with papules and excoriations on the arms and lower legs. This rash began during the time he was placing new insulation in the attic.

The woman complains of a rash with redness and a small amount of blistering on the hands and wrists. There is mild itching, and some painful fissures have formed on the fingertips. The rash developed over a period of several days, beginning with only erythema while she was using a commercial paint-stripping product to remove old paint from interior trim. Although she wore rubber gloves, some of the stripping compound came in contact with her skin by running down into the gloves from the wrist area and through small holes in the fingers.

(1a) What is the most likely cause of the husband's rash? How could this be confirmed?

(1b) What are the most likely causes of the woman's rash?

(1c) How would you treat the skin lesions experienced by these patients?

Description

- **More than 90% of skin lesions caused in the workplace are contact dermatitis.**
- **Lesions of irritant contact dermatitis are localized and the symptoms are generally less severe than those of allergic contact dermatitis.**

In the occupational exposure setting, the most common skin lesions (greater than 90%) are dermatitis due to either contact irritation or contact allergy, with irritant contact dermatitis being reported more frequently.

Irritant contact dermatitis caused by chronic exposure to mild irritants typically begins with erythema and progresses to eczema with exudative vesicles and papules, most often limited to the area of direct contact. Itching, stinging, and burning sensations may be noted—especially with stronger irritants—but are generally not as severe as symptoms of patients who have allergic contact dermatitis. (For a discussion of allergic contact dermatitis, see page 9.)

After days to weeks of chronic irritant exposure, the skin may become lichenified. Painful fissures may develop, along with hyperpigmentation, crusts, and scales. When contact with the offending irritant is discontinued, the rash usually resolves spontaneously in 1 to 3 weeks. Irritant contact dermatitis rarely spreads to areas of the body remote from the site(s) of direct contact.

Cutaneous hardening can develop when patients with irritant contact dermatitis have daily exposure to irritating substances. The skin becomes tough and resistant at the sites of contact, allowing further exposure to the irritant but without reaction. If exposure ceases, however, this protective adaptation is lost rapidly.

Pathophysiology

- **Irritant contact dermatitis is caused by direct action of irritants on the skin.**

Irritant substances cause dermatitis by direct chemical action (i.e., nonimmune-mediated) on contacted components of the skin. Irritants may be acidic substances, which coagulate skin proteins, or alkaline substances, which remove surface lipids. Both types of substances may cause drying and cracking of the skin. Epidermal necrosis with separation of the epidermis from the underlying dermis results in formation of vesicles that contain mainly polymorphonuclear (PMN) leukocytes. Vesicles and bullae with both PMN leukocytes and lymphocytes occur in the upper portion of the dermis.

Common Etiologies

Almost any substance can be a contact irritant (Table 1), although some substances, such as some alcohols, oils, and glycols cause irritant contact dermatitis in only a small percentage of exposed persons. In contrast, strong irritants, such as concentrated mineral acids, alkalies, and amines, cause chemical burns or irritant contact dermatitis in almost everyone exposed. Mild to moderate irritants (e.g., dilute acids, organic hydrocarbon solvents, and some detergents) generally produce irritant dermatitis in only a small percentage of persons after a single contact but will cause a reaction in nearly everyone after prolonged or repeated exposure.

Table 1. Common irritants in the home and workplace*

Home

- Bleaches
- Copper and metal brighteners
- Detergents
- Drain cleaners
- Fertilizers
- Furniture polishes and waxes
- Oven cleaners
- Pesticides
- Pet shampoos
- Rug shampoos
- Scouring pads and powders
- Soaps
- Toilet bowl cleaners
- Window cleaners

Workplace

- Acids and alkalies
- Cleaning products
- Epoxy resins
- Foams (e.g., insulation foams)
- Noncarbon-required (NCR) paper
- Powders
  - Aluminum
  - Calcium silicate
  - Cement
  - Cleaning agents
  - Metallic oxides
- Particles
  - Ore particles in mining
  - Plant particles
  - Plastics, dry
  - Sawdust
  - Wool
- Volatile substances
  - Ammonia
  - Formaldehyde
  - Organic solvents

*Adapted from Robert M. Adams, Occupational skin disease, 2nd edition. Philadelphia: W.B. Saunders Co., 1990.

The young are generally more susceptible to irritant contact dermatitis than adults are because the threshold for skin irritation is low in children, particularly infants. Irritation reactivity gradually lessens after about 8 years of age. During play, children are likely to have skin contact with soils containing hazardous substances or with wooden playground structures that may have been treated with irritating chemicals such as arsenate and pentachlorophenol. The occurrence of skin problems is also common in the elderly. Besides age, personal factors that predispose persons to irritant contact dermatitis include genetic constitution and previous episodes of eczema.

Environmental and physical factors influence the skin's susceptibility to irritant contact dermatitis. Susceptibility is often enhanced by wet work and conditions such as cold and windy weather, low relative humidity, and high temperatures that cause sweating. Some anatomic regions are more sensitive than others. Friction and lacerations or other mechanical skin injury may facilitate the development of irritant contact dermatitis. Occlusion by protective equipment such as gloves provides a humid environment, minimizing evaporation and making the stratum corneum more permeable to chemical substances that come in contact with the skin.

Diagnosis

- **Onset of irritant contact dermatitis tends to be insidious.**

Irritant contact dermatitis is often difficult to differentiate from allergic contact dermatitis. Routine skin biopsy generally is not helpful because the histologic appearance of irritant and allergic contact dermatitis is similar. However, unlike allergic contact dermatitis, irritant contact dermatitis tends to localize at the exposed area and to cause mild itching and more erythema than vesiculation. The onset of irritant contact dermatitis is insidious rather than explosive. Patch testing by, or in consultation with, a dermatologist may be necessary to reach a diagnosis or to exclude allergic contact dermatitis. If fibrous glass is the suspected irritant, skin scrapings suspended in a few drops of 10% potassium hydroxide and examined under a light microscope at low power may reveal glass fibers.

Treatment

- **Removal from exposure is the most important step in treating irritant contact dermatitis.**

The most important step in treatment is to remove the patient, at least temporarily, from further exposure to the offending agent. Substituting less irritating chemicals for the offending substance and correctly using protective materials, such as gloves and barrier creams, may help reduce exposure. During healing, the skin should be protected from other insults such as frequent washing, trauma, wind, and rapid changes in temperature.

Treatment for acute vesicular irritant contact dermatitis includes topical application of wet dressings for 15 to 20 minutes, 3 to 6 times daily. Domeboro's solution (diluted 1:40) or Burow's solution may be used to soak the dressings. Dressings should be discontinued after 2 to 3 days to avoid drying the skin.

Topical application of corticosteroid preparations may be efficacious. A low-potency corticosteroid should be used for mild to moderate skin conditions, with progression to more potent corticosteroids as required (Table 2). Some over-the-counter and prescription topical medications or their excipients can further irritate the skin or provoke allergic contact dermatitis. Administering mild sedatives and antihistamines to relieve itching may also be beneficial.

Clinical signs of secondary bacterial infection include increased erythema and tenderness; development of a yellow, crusting, or purulent exudate; and occasionally, formation of small pustules around the edges of the dermatitis. Infection with monilia has an appearance similar to bacterial infection, except that the exudate is usually white. Infection may be difficult to recognize initially because the serous exudate and erythema of the dermatitis can obscure the signs. Obtaining samples of the exudate for culture and sensitivity before initiating topical or systemic antibiotic therapy is generally advisable.

- **Topical corticosteroids may be useful in cases of irritant contact dermatitis.**

Table 2. Groups of topical corticosteroid products, in order of decreasing potency*

Drug	Trade Name†	% Concentration
Group I		
Betamethasone dipropionate	Diprolene	0.05
Halbertasol propionate	Ultravate	0.05
Clobetasol propionate	Temovate	0.05
Diflorasone diacetate	Psorcon	0.05
Group II		
Amcinonide	Cyclocort	0.1
Betamethasone dipropionate	Diprosone	0.05
Desoximetasone	Topicort	0.25
Diflorasone diacetate	Florone, Maxiflor	0.05
Fluocinolone acetonide	Synalar–HP	0.2
Fluocinonide	Lidex	0.05
Halcinonide	Halog	0.1
Triamcinolone acetonide	Aristocort, Kenalog, etc.	0.5
Group III		
Betamethasone benzoate	Benisone, Uticort	0.025
Betamethasone valerate	Betatrex, Beta-Val	0.1
Desoximetasone	Topicort LP	0.05
Flurandrenolide	Cordran	0.025
Hydrocortisone valerate	Westcort	0.2
Triamcinolone acetonide	Aristocort, Kenalog, etc.	0.1
Group IV		
Betamethasone valerate	Valisone, Reduced Strength	0.01
Clocortolone pivalate	Cloderm	0.1
Fluocinolone acetonide	Fluonid, Flurosyn, Synalar, etc.	0.025
Flurandrenolide	Cordran SP	0.025
Triamcinolone acetonide	Aristocort, Kenalog, Triacet	0.025
Group V		
Alclometasone dipropionate	Aclovate	0.05
Desonide	DesOwen, Tridesilon	0.05
Fluocinolone acetonide	Fluonid, Synalar	0.01
Group VI		
Dexamethasone	Aeroseb-Dex, Decaderm	0.01-0.1
Hydrocortisone	(generic, over-the-counter)	0.25-2.5
Methylprednisolone acetate	Medrol	0.25-1.0

Adapted from RC Cornell and RB Stoughton. The use of topical steroids in psoriasis. Dermatol Clin 1984;2:397-409.

* No significant difference exists among agents in a group. These products come in various forms (i.e., creams, gels, lotions, solutions, and ointments), although some products are not available in all forms.

† Use of trade names is for identification only and does not imply endorsement by the Public Health Service or the U.S. Department of Health and Human Services.

Case 2 — Allergic Contact Dermatitis

You are consulted by a 44-year-old male office worker who has a chief complaint of a rash on his hands and wrists. His company recently relocated from a building where each employee had a private office to an older, renovated building with large bay areas. New wallboard was placed, the area was painted, and new carpet was laid just before the move. Employees now work in cubicles; the patient's cubicle is located in an interior area with no windows. A copying machine is adjacent to his work area.

Since the move, many of the patient's coworkers have been complaining of unpleasant odors, a feeling of fatigue or excessive tiredness, and mild irritation of the eyes, nose, and throat. They associate these symptoms with working in the new area. Although the patient has not noted such symptoms, he does complain of the increased noise and distraction in the new work area; he feels that his rash is somehow related to the new location.

The rash began 5 days ago with itching and redness. It then developed weeping and raised, vesicular lesions that spread from the initial location on the hands to the volar surfaces of the wrists. The patient states that he has a history of reaction to poison ivy, which produces a similar rash, but he has not been in an infested area for the past 2 months. He has no direct contact with industrial cleaning agents or carbonless copy paper in his work. He does have contact with chemicals through his woodworking hobby. He recently built an end table from exotic Japanese woods and has been applying a varnish that a friend brought from Japan.

(2a) Could the patient's rash be due to airborne allergens or irritants in the new office location?

(2b) Could the rash be related to his woodworking hobby?

(2c) What is the most effective treatment for this patient?

Description

- **About 30% of occupational skin disorders are allergic contact dermatitis.**
- **In sensitized persons, inflammation begins about 12 hours after exposure to an allergen.**

Although contact allergens produce sensitization in only a small percentage of exposed persons, allergic contact dermatitis constitutes about 30% of the skin disorders found in the workplace. Once a person has been sensitized to an offending substance, further exposure may result in relatively rapid development of local inflammation with erythema, papule formation, induration, and weeping vesiculation. Inflammation usually begins about 12 hours after exposure; intensity peaks in 50 hours or more. The rash may spread locally around the margins of the original site or to distant sites that did not have contact with the allergen. Potentially, the entire skin surface could become involved (a condition known as erythroderma or exfoliative dermatitis).

Pathophysiology

- **Cross-reactivity with antigenically similar substances can occur with allergic contact dermatitis.**
- **The clinical and histologic appearances of allergic and irritant contact dermatitis are similar.**

Allergic contact dermatitis results from a true allergic (i.e., cell-mediated) sensitization to the offending substance. Cross-reactivity with antigenically similar substances may occur. Initially, during the refractory period, the patient may be exposed without developing a reaction. During the induction phase, which may last from 4 days to several weeks (usually about 14 to 21 days), the development of complete allergic sensitization occurs as the allergen comes in contact with the skin. After the skin is fully sensitized, further contact with the allergen may result in rapid and severe dermal manifestations. When no further contact with the allergen occurs, the patient is in the period of persistence of sensitivity. The level of sensitivity can decrease over time, but sensitization may be lifelong.

Most allergens that cause allergic contact dermatitis have molecular weights of less than 500 daltons. The allergens are haptens rather than complete antigens; they must penetrate the skin and combine with endogenous proteins to form full antigens. Langerhans cells play a key role in then presenting the antigen to T lymphocytes, thereby activating the T cells. The sensitized T cells proliferate in the paracortical regions of the lymph nodes and produce effector and memory lymphocytes that remain in the general circulation. On subsequent contact with the complete antigen, the effector cells release lymphokines that may result in rapid and severe, local inflammation.

Many factors can affect the development of allergic contact dermatitis, including characteristics of the allergen itself, patient factors, and environmental conditions. Allergen factors include the physiochemical nature of the allergen (e.g., lipophilicity, solubility, and inherent sensitizing potency), concentration, total dose that comes in contact with the skin, anatomic site of contact, number and frequency of exposures, and occlusion by clothing or gloves.

The most important predisposing patient factors are a history of irritant contact dermatitis and the presence of an inflammatory skin condition that may promote absorption of the allergen. Irritant dermatitis caused by household cleaning agents on women's hands may continue as allergic nickel dermatitis (from costume jewelry). In addition, age and genetic predisposition can influence the development of allergic contact dermatitis. Persons who have histories of atopic dermatitis have been reported to have *decreased* risk of developing allergic dermatitis but *increased* risk of developing irritant dermatitis.

Common predisposing environmental factors for allergic contact dermatitis are pressure, friction, heat, and prolonged immersion in water (such as occurs during wet work). Relative humidity, ambient temperature, and season of the year also play roles in development of allergic contact dermatitis.

Common Etiologies

Only several hundred of the thousands of chemicals used are known to cause allergic contact dermatitis. With the exception of nickel, cobalt, and some forms of chromium, most metals do not produce sensitization. Strong inorganic alkalies and acids seldom cause allergic reactions. Although a substance's sensitization potential cannot be determined from its chemical structure alone, some chemical classes are more likely to cause allergic contact dermatitis (see Table 3). Aromatic compounds with polar or ionic substituents are typically sensitizing agents (e.g., *p*-aminophenol and hydroquinone used in photographic film developers).

❑ **Aromatic compounds with polar or ionic substituents are potent sensitizing agents.**

In addition, chemicals that are structurally similar to the original sensitizing agent may provoke recall of the specifically sensitized lymphocytes, a phenomenon known as cross-sensitization. For example, persons exposed to p-phenylenediamines used in the rubber industry may react to related substances used in photographic developers and dyes. Persons sensitized to *Rhus* plants such as poison ivy or poison oak may be sensitive to cross-reacting substances found in exotic trees and their derivative products (lacquers, varnishes, and oils).

Table 3. Some chemical groups known to cause allergic contact dermatitis

Aromatic amines	Hydroxyquinolines
Benzothiazoles	Phenolic compounds
Caine-type anesthetics	Phenothiazines
Ethylenediamine compounds	Streptomycin group of antibiotics
Halogenated germicides	Thiurams

Synthetic substances that commonly cause allergic contact dermatitis are rubber products, plastic resins, organic dyes, topical medications, germicidal and biocidal preparations, and various commercial and medication ingredients (Table 4). Natural products can also produce allergic contact dermatitis. Exposure to certain airborne contaminants may also cause allergic contact dermatitis. Airborne contaminants include dichromates in cement dust, rosins used in soldering operations, and sawdust.

Table 4. Common causes of allergic contact dermatitis

Germicides and biocides
- Formaldehyde-releasing compounds
- Parabens
- Quaternary ammonium compounds

Grains
- Barley
- Oat
- Rye
- Wheat

Foods/Spices
- Cardamon
- Carrot
- Chicory
- Coconut
- Coffee
- Endive
- Lettuce
- Potato
- Radish
- Tamarind
- Tumeric
- Vanilla

Medication/product ingredients
- Preservatives
  - Lanolin
  - Thimerosal
- Fragrances and perfumes
  - Balsam of Peru
  - Benzyl alcohol
  - Cinnamic acid derivatives
  - Citronella derivatives

Metals
- Chromium
- Cobalt
- Nickel

Organic dyes
- *p*-Aminoazobenzene
- *p*-Phenylenediamine

Plastic resins
- Epoxies
- Formaldehyde-based acrylics
- Phenolics

Rhus* plants
- Poison ivy
- Poison oak
- Poison sumac

Rubber products
- Antioxidants
- Polymerization accelerators

Topical medications
- Benzocaine
- Neomycin

* For a more complete listing of plants that cause dermatitis see R.M. Adams, Occupational skin disease, 2nd edition, Philadelphia: W.B. Saunders Co., 1990, p. 507-9.

Diagnosis

❑ **Allergic contact dermatitis often spreads to areas remote from the site of contact.**

Allergic contact dermatitis is often misdiagnosed as irritant contact dermatitis. Other conditions to consider in the differential diagnosis are atopic dermatitis, pustular eruptions on the palms and soles, psoriasis, *Herpes simplex* and *Herpes zoster*, insect bites, parasite infestation such as scabies, fungal infections of the feet with idiopathic vesicular reactions, nummular eczema, drug eruptions, and erythema multiforme.

No distinctive features of the lesions facilitate the differentiation of allergic from irritant contact dermatitis. An important diagnostic clue to allergic contact dermatitis is the spread of rash to areas remote from the site of contact; the mucous membranes are usually spared, and the scalp, soles, and palms are often unaffected.

Patch testing (see *Diagnostic Procedures*, page 35) may help differentiate allergic from irritant contact dermatitis. Because the histologic appearance of lesions due to allergic or irritant contact dermatitis is the same, routine skin biopsy is not helpful in their differentiation.

❑ **The clinical and microscopic appearances of skin lesions due to allergic contact dermatitis are the same as those due to irritant contact dermatitis.**

Treatment

At present, there are no satisfactory means of desensitizing humans to allergens. The most important step is to remove the patient from exposure to the offending substance. In the workplace, options such as protective clothing and substitute chemicals should be explored. The therapy for allergic contact dermatitis is the same as that for irritant contact dermatitis (see *Treatment, Irritant Contact Dermatitis*, page 6).

Systemic corticosteroids may be indicated for some patients who have allergic contact dermatitis, especially when large areas of the skin (20% total body surface area or greater) are involved. Short courses of oral corticosteroids, particularly if used for a *Rhus*-induced contact dermatitis, may be given for 2 to 3 weeks (up to 21 days). Corticosteroids administered even for a short period of time should always be delivered in decreasing doses over the course of therapy to prevent adrenal suppression.

❑ **Treatment for allergic contact dermatitis is identical to that for irritant contact dermatitis.**

Case 3 — Photosensitivity Contact Dermatitis

You are consulted by the headmaster of a children's summer camp because of an outbreak of skin rashes in 20 of the campers. No counselors are affected. On examination, the rashes, which are confined to the hands, wrists, and forearms, consist of discrete linear streaks and patches that are hyperpigmented and do not itch.

One of the staff members speculates that the rashes are caused by contact with an epoxy glue used in building a model. However, only two of the children who have rashes have been involved in this activity. All the affected children had participated in a craft class in which they made lime sachets by puncturing lime skins and inserting sprigs of cloves over the surfaces of the limes. During the class, they also prepared gift cards from recycled paper. While the children attended to these activities, the counselors were engaged in planning an outdoor activity that was to follow the craft session.

(3a) What causes of the children's dermatitis might be considered, given the rural location and nature of camp activities?

(3b) What treatment would you recommend?

Description

- **Photosensitivity reactions occur mainly on sun-exposed areas of the body.**
- **Photoallergic reactions are immune-mediated responses; phototoxic reactions are not.**

Photosensitivity contact dermatitis occurs mainly on sun-exposed areas such as the face, upper chest, posterior portion of the neck, extensor surfaces of the forearms, dorsum of the hands and feet, and anterior surfaces of the lower legs. Areas of the skin normally covered by jewelry and clothing are spared, as are eyelids, areas under the chin, and upper portions of the ears covered by hair. Photosensitivity contact dermatitis may be the result of phototoxicity or photoallergy.

Lesions of phototoxic and photoallergic contact dermatitis resemble those of irritant and allergic contact dermatitis. They have been described as discrete, confluent, polymorphous linear streaks and patches that are macular and nonpruritic. The patient may experience a stinging or burning sensation of the skin, typically beginning shortly after exposure to sunlight and resolving rapidly when the skin is shaded. Lichenification and hyperpigmentation may occur, and the lesions may persist for months or years. In some cases, widespread involvement of the skin develops later. The photoallergic response usually occurs in only a small number of persons who have been previously sensitized to the photoactive agent.

Pathophysiology

- **Sunlight can cause formation of the agents that result in photosensitivity contact dermatitis.**

The mechanisms of photosensitivity contact dermatitis are broadly analogous to the mechanisms of irritant and allergic contact dermatitis except for the added requirement of appropriate ultraviolet (UV) radiation (i.e., wavelengths of 315 to 400 nanometers, known as UV-A). The agent that provokes the irritant or allergic response is formed after its precursor has been exposed to UV-A.

In phototoxicity, the excited state of the agent produced during irradiation is thought to lead to oxidation of cellular components or to allow binding of the agent with nucleic acids. In photoallergy, the initial reaction of the topical agent with UV-A forms either an excited molecule that can bind with protein to form a complete allergen or a product that is itself a strong contact allergen.

Common Etiologies

- **Many topical products can produce photosensitivity dermatitis.**

Many products that cause photosensitivity dermatitis are applied topically. Common examples are lotions containing fragrances; suntanning products with ultraviolet absorbers such as 6-methylcoumarin, homosalicylate, or *p*-aminobenzoic acid (PABA); and aftershave lotions containing musk ambrette. Germicides in soaps and detergents may also cause photosensitivity dermatitis. A major epidemic of allergic contact dermatitis occurred in Great Britain in 1960 after the introduction of two soaps that contained tetrachlorosalicylanilide, a photoactive antibacterial agent.

Certain systemically administered medications have caused photoallergic drug reactions. Examples include nalidixic acid, phenothiazines, sulfonamides, sulfonylureas, tetracyclines, and thiazide diuretics. Pharmacists, nurses, and others who routinely have skin contact with these drugs are prone to photosensitivity dermatitis.

Plants such as celery and citrus fruits have caused phototoxic dermatitis in persons who handle them extensively; farm workers are particularly susceptible. Contact with oil released from lime skins or with coal tar and pitch has resulted in phototoxic dermatitis, especially in lightly pigmented persons.

Psoralens, which are photoactive and can cause phototoxic dermatitis, are also used therapeutically in the treatment of psoriasis. In PUVA (psoralen plus UV-A radiation) treatment, a psoralen is painted on the affected skin or given systemically to patients who are then exposed to UV radiation. The photoadduct that is formed between the psoralen and DNA serves to slow the rate of the psoriatic overgrowth.

- **Psoralens, which can cause phototoxic dermatitis, are useful in the treatment of psoriasis.**

Diagnosis

Photoallergy from chemical contact must be differentiated from polymorphous light eruption, systemic lupus erythematosus, pellagra, dermatomyositis, porphyria, allergic contact dermatitis and photoallergic drug reaction. A thorough history of medication treatment will usually rule out photoallergic drug reaction.

Photopatch testing may be useful in confirming the diagnosis, but results of photopatch testing are often difficult to interpret and are best left to dermatologists with specialized equipment and knowledge in this field.

- **A thorough drug history will usually rule out photoallergic drug reactions.**

Treatment

The treatment for phototoxic or photoallergic dermatitis is the same as the treatment for irritant and allergic contact dermatitis (see pages 6 and 13). Identifying the offending agent and counseling the patient to avoid further exposure to it are the most important interventions. When the photosensitizing agent cannot be avoided, limiting sunlight exposure and wearing protective clothing, such as hats, gloves, long-sleeved garments, socks, and shoes, may help. Sunscreens may be used if reaction or cross-sensitivity between the causative agent and components in the sunscreen is not a possibility.

- **Treatment for phototoxic and photoallergic dermatitis is the same as that for irritant and allergic contact dermatitis.**

Case 4 — Chloracne

A 42-year-old man consults you because of a persistent skin condition that he feels resembles the cosmetically displeasing acne he had as a teenager. His present skin condition consists of pale yellowish, cystic lesions and comedones localized on the face, below and lateral to the eyes, and behind the ears. Similar lesions are present on the cheeks, forehead, and neck; a few are present on the buttocks, where, according to the patient, he never had lesions with his prior affliction. He also complains of moderately severe itching.

History reveals no changes in diet, and the patient is not taking medications. For the last 15 years, the patient has worked for a local utility company. His most recent job duties have included replacing the heat exchange fluids in transformers. He first noted the rash about a month ago; he is not certain whether the rash appeared before or after he began this activity.

(4a) Is the skin condition described by the patient consistent with a reactivation of acne vulgaris? What other causes should be considered?

(4b) What therapy would you recommend?

Description

- **Chloracne is rare and is usually due to occupational exposure to chloracnegenic agents.**
- **Chloracne may be an indication of systemic toxicity.**

Environmental acne is a variety of acne venenata typically caused by industrial chemicals. It may result from contact with petroleum and its derivatives (oil acne), coal tar products (coa-tar-pitch acne), and halogenated aromatic hydrocarbons (chloracne). Environmental acne may also be caused by certain physical, mechanical, and biologic agents. Although the occurrence of chloracne is rare (probably fewer than 4000 cases worldwide), it is of great concern because it is an extremely refractory acne and because it may be indicative of systemic toxicity by a highly toxic chemical.

The lesions of chloracne consist of straw-colored cysts, numerous comedones, milia, and papules. The lesions are located on the face (especially at "crow's feet" and below and to the outside of the eyes [malar crescent]), neck, earlobes, shoulders, abdomen, legs, buttocks, and genitalia. The nose is often spared. With severe chloracne, all the follicles in an area may be involved, resulting in a rather bizarre "pebbled" appearance. Pruritus is common and occurs in about 50% of chloracne cases.

Pathophysiology

- **Chloracne is often refractory to treatment.**

Onset of disease is typically delayed 2 to 4 weeks after exposure to a chloracnegenic agent. The first changes are a thickening of the follicular epithelium, development of comedones, and a slow disappearance of the sebaceous glands as they are replaced by keratinous cysts. Initially, inflammation is uncommon; inflammatory lesions with larger cysts and abscesses are later developments. Severe scarring may occur. Increased fragility of the skin, hypertrichosis, widespread follicular hyperkeratosis, or hyperpigmentation may develop. A brownish discoloration of the nails, swollen eyelids, and conjunctivitis or discharge may be present in some patients.

With no additional exposure, the disease will first progress, then regress over a 4- to 6-month period. A few cases of chloracne have persisted for 30 years or more after contact with the chloracnegenic agent has ceased.

Common Etiologies

- **Chlorinated aromatic hydrocarbons cause chloracne.**

Many chlorinated aromatic hydrocarbon compounds used in the workplace can cause chloracne. These compounds include chlorinated naphthalenes, polychlorinated biphenyls (PCBs), polybrominated biphenyls (PBBs), dioxins, polychlorinated dibenzofurans, pentachlorophenol, azobenzenes, and azoxybenzenes. (See *Case Studies in Environmental Medicine: Polychlorinated Biphenyl (PCB) Toxicity, Case Studies in Environmental Medicine: Dioxin Toxicity, and Case Studiies in Environmental Medicine: Pentachlorophenol Toxicity.)*

Diagnosis

Chloracne must be differentiated from oil acne or folliculitis due to exposure to grease and oils; acne vulgaris; acne cosmetica from heavy cosmetic use; acne mechanica from local pressure and friction; acne medicamentosa from medications such as corticosteroids, hormonal preparations, phenytoin, iodides (e.g., in kelp tablets), bromides and solar elastosis with comedones.

❑ Chloracne must be differentiated from other more common acnes.

A history of exposure to agents known to cause chloracne and the typical appearance of the rash on physical examination are usually sufficient for diagnosis. Chloracne may be distinguished from acne vulgaris by the distribution of the lesions, age at onset, and morphology. Chloracne lesions typically affect the face, neck, earlobes, shoulders, abdomen, legs, buttocks, and genitalia, whereas lesions of acne vulgaris are found primarily on the face, neck, chest, and back (down to the waist). Chloracne can appear at any age, whereas acne vulgaris is seen most often in patients aged 13 to 26 years. Chloracne lesions consist of straw-colored cysts, numerous comedones, milia, and papules; whereas the lesions of acne vulgaris are typically comedones, papules, pustules, and scars.

Histologic examination of cysts may show typical changes, but the usefulness of biopsy in establishing the diagnosis is questionable. Associated noncutaneous conditions found in some patients who have chloracne include hepatotoxicity, porphyria cutanea tarda, and peripheral neuropathies.

Treatment

Primary interventions are prevention of exposure to chloracnegenic chemicals and good hygiene because a satisfactory treatment regimen cannot be found in many cases. Administration of oral antibiotics and acne surgery have been of limited success. Retinoic acid (vitamin A) preparations or 13-cis-isoretinoic acid (Accutane) have been successful in carefully selected patients. (Note: Accutane is a known teratogen and should be used cautiously.) Injecting inflamed lesions with dilute triamcinolone, a glucocorticoid, may be helpful, as may dermabrasion for severe scarring.

❑ Removal from exposure to chloracnegenic agents is the most important treatment for chloracne.

Case 5 — Pigment Alterations

Fifteen children from a local school are referred by the school nurse for evaluation of skin lesions. The lesions consist of decreased pigmentation in a scattered distribution. Two of the children have histories of itchy, weeping, vesicular rash on the neck and face that cleared before the pigment changes became noticeable. A public health evaluation of the drinking water and food served at the school has not revealed toxic or infectious agents. The school is located near a chemical manufacturing facility, in which the parents of several children work, including the parents of the two children who have histories of vesicular rash.

(5a) Could the nearby manufacturing facility be associated with the decreased pigmentation noted in the children in this case?

(5b) How could you investigate this possibility?

(5c) What treatment options are available for persistent hypopigmentation involving large areas of the skin?

Description

- **Pigment changes are usually associated with post-inflammatory effects from physical or chemical agents.**

A variety of physical and chemical agents may affect the color of the skin. Insults to the skin may cause either increased pigmentation (hyperpigmentation), decreased pigmentation (hypopigmentation), or both in contiguous areas (dyschromia). Inflammation, which may be subclinical and not apparent, usually precedes pigment alterations. Postinflammatory reaction (e.g., to contact dermatitis) is the most common cause of increased pigmentation, although pigment loss may also occur.

Pathophysiology

- **Hypopigmentation is caused by damage to the melanocyte or through inhibition of melanin synthesis.**
- **Hyperpigmentation is often caused by nonspecific skin damage that leads to melanin or hemosiderin accumulation.**

In hypopigmentation, depigmentation probably occurs either by damage to the melanocyte, which leads to cell distortion and death, or through inhibition of melanin synthesis by the offending substance. It may be significant that industrial compounds that cause hypopigmentation (Table 5) are structurally similar to tyrosine, the building block of melanin. In industrially related hypopigmentation (leukoderma), the hands, wrists, and forearms invariably are affected; symmetry is usual. Depigmentation may also appear in body sites remote from the chemical contact (e.g., axillae, genitalia, and torso). The process of depigmentation usually takes 2 to 4 weeks and may require up to 6 months of repeated contact to become visible. The fact that many exposed workers do not lose pigment indicates that host factors are important in susceptibility.

Table 5. Compounds known to cause hypopigmentation

o-Benzylchlorophenol (antiseptic)
p-Butylphenol (used in the manufacture of varnish and lacquer resins, as an antioxidant in soaps, and as a motor oil additive)
p-Cresol (disinfectant)
Hydroquinone and its monoethyl and monobenzyl ethers (used in black-and-white photoprocessing, in skin lighteners, and as antioxidants in synthetic rubbers)
o-Phenylphenol (used as an agricultural fungicide, disinfectant, and in the rubber industry)
Pyrocatechol (topical antiseptic)
p-Tertiary butylcatechol (astringent)

Hyperpigmentation (also known also as melanosis or melanoderma) is due to accumulation of melanin from damaged melanocytes or to deposition of hemosiderin from extravasation of erythrocytes in the dermis. Another possible mechanism is overproduction of melanin by melanocytes in response to the offending agent. Hyperpigmentation is more likely to occur in dark-complexioned persons and can persist for years.

Common Etiologies

The cause of hypopigmentation is contact with alkylphenols (see Table 5, page 24), skin damage due to chemical and thermal burns, or blunt or repeated trauma to the skin. Hyperpigmentation typically follows a bout of dermatitis or other episode of inflammation. Coal tar pitch, creosote, and various aromatic chlorinated hydrocarbons are a few of the compounds that can stimulate overproduction of melanin. UV radiation-induced stimulation of melanin synthesis (tanning) is the most common cause of hyperpigmentation in dark-complexioned persons.

Diagnosis

Chemically induced hypopigmentation is indistinguishable from idiopathic vitiligo. Vitiligo affects about 1% of the general population and may be associated with autoimmune or endocrine abnormalities. Hypopigmentation must also be differentiated from depigmentation due to tissue destruction by chemical or thermal burns.

Hyperpigmentation should not be confused with birthmarks or direct skin staining or discoloration from contact with substances such as heavy metals (e.g., silver salts), nitrosylated compounds (e.g., nitric acid or dinitrophenol), derivatives of coal distillation (e.g., tar, pitch, and asphalt), and coal dust.

In most cases, the patient's history and physical examination are sufficient to diagnose cases of pigment alterations. The loss of melanin in light-complexioned persons can be detected by failure of the skin to fluoresce under a Wood's lamp.

- **Hypopigmentation may be the result of environmental exposures or of idiopathic vitiligo.**
- **Skin staining and birthmarks can be misdiagnosed as hyperpigmentation.**

Treatment

No effective treatment exists to reverse pigment changes. Hypopigmentation may last months to years after contact with the offending substance is discontinued, or it may be permanent. Depigmented skin should be protected from sunlight. Small depigmented areas may be camouflaged with agents such as Covermask, Dy-O-Derm, or Dermablend. Oral administration of psoralens and carefully graded UV radiation exposure (PUVA treatment) may be attempted if hypopigmentation involves large areas of skin.

In patients who have hyperpigmentation, worsening of the condition can be prevented by using sunscreens and covering affected areas

- **Hypopigmented and hyperpigmented areas should be protected from sunlight.**

Case 6 — Contact Urticaria

A 35-year-old woman consults you because of episodes of generalized hives that develop about 20 minutes after she uses certain brands of shampoo. The hives are preceded by sensations of itching, burning, and stinging of the skin on the scalp, upper face, and posterior aspect of the neck. The patient also experiences redness and tearing of the eyes, clear rhinorrhea, and nausea. She relates that a similar constellation of symptoms occurred after she applied an over-the-counter topical pain-relieving ointment for sunburn. A mild eczematous rash has been present on her forehead and posterior neck for about 6 weeks.

(6a) What is the most likely cause of the patient's complaints?

(6b) What evaluation and testing might be helpful?

(6c) What treatment will most likely be effective?

Description

- **Contact urticaria is a skin reaction that appears immediately after contact with the offending agent.**

Contact urticaria is a localized wheal-and-flare response (hives) that develops almost immediately (a few minutes to about 1 hour) after direct contact with the eliciting agent. Many afflicted patients complain of skin sensations such as itching, burning, or tingling. Symptoms typically disappear within 24 hours.

Pathophysiology

- **Contact urticaria may be due to immunologic-, nonimmunologic-, or uncertain-mediated mechanisms.**
- **Anaphylactic reactions may occur in patients who have contact urticaria syndrome.**

Contact urticaria may be mediated by mechanisms classified as immunologic (allergic), nonimmunologic (nonallergic), or uncertain. Nonimmunologic urticaria, the most common type of contact urticaria, is caused by a direct action of the offending substance on the skin vasculature and a nonimmunologic release of vasoactive substances such as bradykinin, histamines, or other inflammatory mediators. The reaction remains localized.

Immunologic contact urticaria is an immediate allergic reaction in persons who have previously become sensitized to the offending agent. Parts of the skin that are remote from the contact site may be affected. The vasoactive effects in the immunologic form of contact urticaria are caused by an IgE-mediated reaction. The resulting erythema and edema are elicited mainly by histamines released from mast cells. Activation of the complement cascade and generation of anaphylatoxins can result in systemic effects (contact urticaria syndrome) in which the typical rash is accompanied by symptoms of asthma, rhinitis, conjunctivitis, orolaryngeal effects (itching and tingling sensations or edema of the lips, tongue, and mouth; or throat irritation), or gastrointestinal signs and symptoms. In rare cases, patients who have contact urticaria syndrome have experienced otherwise unexplained attacks of vascular collapse (anaphylactoid reactions).

The cause of the third type of contact urticaria is uncertain but includes both allergic and nonallergic mechanisms. Formaldehyde is an example of an urticant that has features of both types.

Common Etiologies

- **Latex rubber gloves are a common cause of immunologic contact urticaria.**

Immunologic contact urticaria is usually caused by proteins or protein complexes. It may also be caused by a wide variety of common chemicals, medications, cosmetics, and other agents (Table 6). The rubber in latex gloves is a common cause of contact urticaria among healthcare professionals, as is the cornstarch used in the gloves. Food-

stuffs are also a common cause of contact urticaria. The orolaryngeal area is a site where immediate reactions are provoked by food allergens, most often among atopic persons.

Table 6. Some substances that cause allergic contact urticaria

Animal products	**Foods**	**Plant products**
dander	eggs	henna
hair	flour	latex rubber
saliva	fruits & vegetables	papain
serum	meats	strawberries
	milk	woods
Common Chemicals	nuts	
ammonia	seafood	**Textiles**
alcohol	spices	silk
parabens		wool
polyethylene glycol	**Medications**	
	bacitracin	**Miscellaneous**
Cosmetics	cephalosporins	acrylic monomer
hair products	chloramphenicol	epoxy resin
nail polish	gentamicin	formaldehyde
perfumes	neomycin	nylon
	salicylic acid	seminal fluid

Nonimmunologic contact urticaria has been provoked by contact with substances as diverse as acids (acetic, benzoic, butyric, cinnamic, sorbic), alcohols (ethyl and butyl), balsam of Peru, benzocaine, cinnamic aldehyde, cobalt chloride, dimethylsulfoxide, formaldehyde, witch hazel, sodium benzoate, and esters of nicotinic acid. Cold temperatures can also cause nonimmunologic contact urticaria.

Uncertain mechanism-mediated contact urticaria has been associated with exposure to ammonium persulfate, which is used to boost peroxide hair bleaches to achieve a platinum-blond effect. Sunlight, which can produce rapid development of a wheal-and-flare reaction in exposed areas, and aquagenic agents (water, saline, or the patient's own perspiration) are also associated with uncertain mechanism-mediated contact urticaria.

Diagnosis

Nonimmunologic contact urticaria must be differentiated from allergic contact urticaria and other forms of urticaria. The most important factor in making the correct diagnosis is taking a careful history of the relationship between possible exposures and development of symptoms. In cases of chronic urticaria, a clear cause is seldom identified.

❑ **A clear cause is seldom identified in cases of chronic contact urticaria.**

Patch or scratch/prick tests may be used with suspected etiologic agents. These tests should be used initially on normal areas of skin,

then on involved skin (previously or currently affected) only when no reaction occurs on normal skin. Testing should be done by, or in consultation with, a dermatologist; resuscitation equipment and medications should be available in case a severe anaphylactoid reaction results.

Treatment

❑ **Antihistamines can alleviate symptoms of urticaria.**

Chlorpheniramine-like antihistamines are of value in treating urticaria. The newer agents that have less sedative effects, such as terfenadine (Seldane) and astemizole (Hismanal), are not as efficacious. (Note: Seldane and Hismanal are contraindicated in patients who are taking ketoconazole, itraconazole, erythromycin, or other medications known to impair the metabolism of Seldane or Hismanal, and in patients who have significant hepatic dysfunction.)

Nonsteroidal anti-inflammatory medications have proved useful in certain cases of nonimmune urticaria; however, they may cause anaphylaxis in patients who have immune urticaria, especially patients who exhibit the triad of asthma, nasal polyps, and rhinitis. These patients should be cautioned about the use of nonsteroidal anti-inflammatory agents. All patients suffering from urticaria should be advised to avoid further contact with the eliciting substance.

Case 7 — Malignant Neoplasms

A couple in their 60s who are native to a nearby rural area consult you because of the insidious development of a variety of skin lesions over the past 2 years. Both have hyperkeratotic lesions on the palms and the soles of the feet, as well as mottled-appearing hyperpigmented areas on the temples and neck. The man has a lesion on the right cheek that appears to be a basal cell carcinoma. Both patients complain of numbness and tingling in the feet and a general feeling of fatigue.

(7a) Assuming that a single agent is responsible for the constellation of complaints of the couple, what sources should be investigated?

(7b) What treatment would you recommend?

Description

- **Skin cancer is the most common neoplasm in adults in the United States.**
- **Sunlight, either alone or in conjunction with other agents, is a major contributing factor to skin cancer.**
- **Reasons for the 700% increase in malignant melanoma in the past 60 years have not been well established.**
- **PAHs and inorganic arsenic are well-known causes of cancerous skin lesions.**

Cancer of the skin is the most common neoplasm among adults in the United States. More than 500,000 new cases of nonmelanoma skin cancer and about 28,000 cases of melanoma occur annually. Skin cancers associated with environmental factors include basal cell carcinoma, squamous cell carcinoma, malignant melanoma, and Bowen's disease (intraepidermal squamous cell carcinoma). Pre-nonmelanoma skin cancers, such as actinic keratoses, can also be induced by environmental factors.

Sunlight, either alone or in conjunction with other agents, plays an important role in the development of most skin cancers, especially malignant melanoma. The incidence of melanoma has increased more than 700% in the past 60 years. If the incidence continues to increase at the present rate, within the next decade a person's lifetime risk of developing melanoma will be approximately 1% (i.e., 1 case of melanoma per 100 persons). The reasons for this increased risk have not been well established but may be related to ozone depletion in the upper atmosphere; increased recreational sun exposure, especially early in life; increased use of industrial chemicals; and increased air pollution.

The usual wavelength in sunlight that causes skin cancers is 280 to 315 nanometers (UV-B). This range is capable of producing direct photochemical damage to the skin (e.g., alterations in DNA and other cellular constituents). UV-B also reacts with photoactive exogenous chemicals in or on the skin, causing them to absorb UV radiation and initiate or accelerate an adverse reaction in normal tissue. Industrial contaminants and air pollutants often contain photoactive chemicals, which can act as photosensitizers, additive carcinogens, or promotors.

The first association between occupational or environmental chemicals and malignancy was noted in 1775 by Percival Pott who reported a high incidence of scrotal cancer among London's chimney sweeps. Years later, it was discovered that the cancers were caused by exposure to certain polynuclear aromatic hydrocarbons (PAHs). PAHs are found in soot, pitch, creosote, petroleum, and oils such as cutting oil, mineral oil, and shale oil. (See *Case Studies in Environmental Medicine: Polynuclear Aromatic Hydrocarbon [PAH] Toxicity.*) Other chemicals found to be associated with skin tumors include phenolic compounds, aliphatic hydrocarbons, and inorganic arsenic compounds.

Inorganic arsenic compounds are known to cause a variety of skin lesions, including malignant neoplasms. Initial dermal manifestations of arsenic exposure may be mild erythema and hyperhidrosis of the palms and soles, followed by development of slightly raised, firm, generally symmetrical punctate keratoses. White-colored, nonraised hyper-keratoses may also develop on the ankles, shins, and dorsum of the hands. A diffuse hyperpigmentation of the skin interspersed with white, somewhat atrophic macules ("raindrops on a dusty road"

appearance) may also be seen. Basal cell and squamous cell carcinomas may then develop. (See *Case Studies in Environmental Medicine: Arsenic Toxicity.*)

Bowen's disease, a squamous cell carcinoma, may arise spontaneously in situ or may develop after chronic exposure to inorganic arsenic or other chemicals. Bowen's disease consists of randomly distributed, sharply demarcated, erythematous, scaling lesions that range in size from a few millimeters up to 1 to 2 centimeters in diameter. The lesions grow slowly and rarely metastasize.

Pathophysiology

Many chemical substances associated with malignant neoplasia are thought to interact directly with cellular macromolecules, resulting in neoplastic transformation of the affected cell. In some cases, absorbed chemicals are converted by skin enzymes (specifically, aryl hydrocarbon hydroxylases) to forms that then combine with DNA and other cellular constituents. Arsenic is thought to inhibit the enzymes involved in DNA replication and repair.

Some chemical agents act either concomitantly (cocarcinogens) or serially (promoters) to cause neoplastic transformation. In many cases, precancerous lesions, such as actinic or arsenical keratoses and tar warts, may precede the development of frank cancerous lesions. A latency period of several decades may lapse between exposure to a carcinogen and appearance of a cancerous lesion.

❑ **The latency period for development of cancerous lesions can be 20 years or more.**

Common Etiologies

Certain chemical agents, such as PAHs and inorganic arsenic, are known to cause skin cancer. Nonchemical agents that may cause malignant neoplasms include sunlight, ionizing radiation, and physical trauma. Sunlight is the most important cause of malignant melanoma.

❑ **Sunlight is the most important cause of malignant melanoma.**

Diagnosis

All potentially cancerous skin lesions must be differentiated from benign lesions. Suspected malignant skin lesions are diagnosed most accurately by histologic examination of excisional biopsies. A punch biopsy of suspect lesions may also be performed.

❑ **Diagnosis of a malignant skin lesion requires a biopsy.**

Treatment of skin cancer depends on whether the cancer is localized or is metastasizing.

Prevention is the first line of defense for skin cancer. Avoiding overexposure to sunlight is most important. Protection from UV radiation can be accomplished by wearing tightly woven clothing and wide-brimmed hats and by applying sunscreens as absorbers. Sunscreens, which contain p-aminobenzoic acid (PABA) derivatives to absorb UV rays, can provide sun-protective factors (SPFs) ranging from 2 to 50 or more. An SPF of 15 allows most persons to remain out of doors for 5 hours before developing minimal erythema. Light-complexioned persons, persons of Celtic origin (i.e., Scotch, Irish, Welsh), and those with certain conditions (e.g., albinism, xeroderma pigmentosum, and erythropoietic protoporphyria) appear to be at increased risk for developing skin cancer. These sensitive populations may require more potent sunscreens.

Surgical excision and radiation are the most common treatment modalities for localized malignant skin lesions. All excised tissue should be sent for histologic examination to confirm the diagnosis and to be certain that an adequate margin of normal skin was removed. Surveillance for the development of further skin cancers should be continued. The treatment of metastasizing skin cancers or lesions with extensive local infiltration is beyond the scope of this review. Patients who have malignant tumors should be referred to, or treated in consultation with, a physician knowledgeable in cancer therapy.

Diagnostic Procedures

Obtaining and recording a detailed history of exposures (workplace, home, and environment) is essential in diagnosing skin disease. Besides physical examination, several special techniques may aid in the diagnosis of skin lesions. These include patch tests to detect contact allergy, skin biopsy, cultures, and microscopic scrapings of skin to detect yeasts, fungi, parasites, and fibrous glass. Referring patients to, or consulting with, a dermatologist who can perform or interpret dermatologic diagnostic testing, may be advisable.

Patch Testing

❑ **Patch testing can help differentiate allergic from other forms of dermatitis.**

Patch testing is frequently used to differentiate between allergic contact dermatitis and other forms of dermatitis. The presence of a delayed hypersensitivity reaction to an offending substance can be determined by placing a suitably prepared, nonirritating amount of a sample on the skin (usually on the back) under a chamber or impervious bandage (patch). If an eczematous dermatitis lesion develops under the patch during the 48 hours after application, allergy to the test substance or to an antigenically similar cross-reacting substance can be inferred. If no reaction is evident, the patches are removed, and the sites are reexamined for delayed reaction at 72 and 96 hours after application.

Interpretation of patch testing is often difficult, and it is usually recommended that the testing be carried out in specialized centers or by consultants who routinely do patch testing. If no response is provoked, it does not mean unequivocally that the patient is not allergic. For example, if an offending or cross-reacting substance was not included, or was not applied in proper concentration, a false-negative result will occur.

Complications of patch testing include the "angry back syndrome," in which the patient's entire back becomes edematous and erythematous. Flare-up of previously existing eczema can also occur, especially when testing materials are not obtained from standard commercial sources. Even local response to the test substance may be extensive, causing patient discomfort. Patch testing itself can result in allergic sensitization to a substance to which the patient was not allergic previously, although this is a rare occurrence. Infections, scarring, and pigment alterations may also be complications of patch testing.

Photopatch Testing

❑ **Photopatch testing may help reveal the cause of photosensitivity dermatitis.**

When photosensitivity dermatitis is suspected, a combination of chemical patch testing and special light exposure may reveal the cause. Duplicate patches are used; one set is covered, and the other

set is exposed to a measured amount of UV radiation. There are several difficulties in the performance and interpretation of photopatch testing, and it should be performed by practitioners who have experience and the requisite special equipment.

Skin Biopsy

- **Skin biopsy is not helpful in differentiating allergic contact dermatitis and irritant contact dermatitis.**

The appropriate skin biopsy (punch biopsy or excision of the lesion) usually can be performed under local anesthesia by experienced practitioners in an outpatient setting. Microscopic examination of the specimens obtained can allow differentiation between benign and malignant skin conditions. Irritant and allergic contact dermatitis cannot be readily differentiated on routine skin biopsy.

Other Diagnostic Procedures

- **Skin scrapings, UV-light examinations, cultures, and serologic testing are diagnostic tools used for various skin lesions.**

Other procedures include skin scrapings, UV-light examinations, cultures, and serologic testing.

Skin scrapings can be used to look for fungal hyphae, for strands of fiberglass in suspected fibrous glass dermatitis, or for scabies mites and eggs. A Wood's lamp, which produces UV radiation, can be used to examine suspected areas of hypopigmentation in light-complexioned persons. Areas deficient in melanin will not fluoresce under UV light, whereas areas of skin with normal melanin content will fluoresce.

Bacterial, viral, or fungal cultures may be indicated if dermal infections are considered in the differential diagnosis. Crusts, when present, should be lifted with a scalpel blade before swabbing the lesion with a sterile cotton-tipped applicator to obtain material for bacterial cultures. Fungi may be collected for culture media by gently scraping the skin with a sterile scalpel blade. Viral cultures from skin lesions require specialized laboratory facilities. Viruses can be collected for special media by unroofing lesions and swabbing with a sterile cotton-tipped applicator.

Patients who have immunologic-mediated contact urticaria may be evaluated by serologic testing. Protein electrophoresis and measurement of circulating IgE may be useful.

Suggested Reading List

General

Adams RM. Occupational skin disease. 2nd ed. Philadelphia: W.B. Saunders Co., 1990.

Maibach HI, ed. Occupational and industrial dermatology. 2nd ed. Chicago: Year Book Medical Publishers, Inc., 1987.

Mathias CGT. Occupational dermatoses. In: Zenz C, ed. Occupational medicine: principles and practical applications. 2nd ed. Chicago: Yearbook Medical Publishers, Inc., 1988:132-65.

Raffle PAB, Lee WR, McCallum RI, Murray R, eds. Occupational diseases of the skin. In: Hunter's diseases of occupations. Boston: Little, Brown & Company, 1987:917-48.

Rosenstock L, Cullen MR. Skin disease. In: Clinical occupational medicine. Philadelphia: W.B. Saunders Co., 1986:147-73.

Rycroft RJG. Occupational dermatoses among office personnel. Occup Med 1986;1:323-8.

Seidenari S, Manzini BM, Danese P, Motolese A. Patch and prick test study of 593 healthy subjects. Contact Dermatitis 1990; 23:162-7.

Shama SK. Occupational skin disorders, In: McCunney RJ, ed. Handbook of occupational medicine. Boston: Little, Brown & Company,1988:216-35.

Suskind RR. Environment and the skin. Med Clin North Am 1990;74:307-24.

Irritant and Allergic Contact Dermatitis

Abel EA, Wood GS. Mechanisms in contact dermatitis. Clin Rev Allergy 1986;4:339-52.

Dahl MV. Chronic irritant contact dermatitis: mechanisms, variables, and differentiation from other forms of contact dermatitis. Adv Dermatol 1988;3:261-75.

Lammintausta K, Maibach HI. Exogenous and endogenous factors in skin irritation. Int J Dermatol 1988;27:213-22.

Photosensitivity Dermatitis

Emmett EA. Drug photoallergy. Int J Dermatol 1978;17:370-9.

Emmet EA. Evaluation of the photosensitive patient. Dermatol Clin 1986;4:195-202.

Gross TP, Ratner L, de Rodriquez O, et al. An outbreak of phototoxic dermatitis due to limes. Am J Epidemiol 1987;125:509-14.

Harber LC, Bickers DR. Photosensitivity diseases. Philadelphia: W.B. Saunders Co., 1981.

Chloracne

American Medical Association. The Health effects of "Agent Orange" and polychlorinated dioxin contaminants: an update, 1984. Chicago: American Medical Association, 1984.

Taylor JS. Environmental chloracne: update and overview. Ann NY Acad Sci 1979;320:295-307.

Tindall JP. Chloracne and chloracnegens. J Am Acad Derm 1985;13:539-88.

Zugerman C. Chloracne. Clinical manifestations and etiology. Dermatol Clin 1990;8:209-13.

Pigment Abnormalities

Gellin GA, Maibach HI, Misiaszek MH, Ring M. Detection of environmental depigmenting substances. Contact Dermatitis 1979;5:201-13.

Gian C, Lu MY, Wang XS, et al. Epidemiologic study on the skin lesions of workers exposed to N,N'-methylene-bis-(2-amino-1,3,4-thiadiazole) and of children in the neighborhood of the factory. Scand J Work Environ Health 1985;11 Suppl 4:55-9.

Ortonne J-P, Mosher DB, Fitzpatrick TB. Chemical hypomelanosis. In: Ortonne J-P, Mosher DB (eds). Vitiligo and other hypomelanoses of hair and skin. New York: Plenum Medical Book Co., 1983: 479-508.

Carcinogenesis

Austin DF, Reynolds P. Occupation and malignant melanoma of the skin. In Gallagher RP (ed). Epidemiology of malignant melanoma. Recent results in cancer research. Vol 102. Berlin: Springer-Verlag, 1986: 98-107.

Callen J, Headington J. Bowen's and non-Bowen's squamous intraepidermal neoplasia of the skin. Arch Dermatol 1980;116:422-6.

Emmett EA. Occupational skin cancers. Occup Med: State Art Rev 1987;2:165-77.

Epstein JH. Photocarcinogenesis: A review. Natl Cancer Inst Monogr 1977;50:13.

Shannon RL, Strayer DS. Arsenic-induced skin toxicity. Hum Toxicol 1989;8:99-104.

Van der Leun JC. UV carcinogenesis. Photochem Photobiol 1984;39:861-8.

Sources of Information

More information on skin lesions and treating and managing cases involving skin lesions due to environmental exposure can be obtained from ATSDR, your state and local health departments, and university medical centers. *Case Studies in Environmental Medicine: Skin Lesions and Environmental Exposures—Rash Decisions* is one of a series. To obtain other publications in this series, please use the order form on the inside back cover. For clinical inquiries, contact ATSDR, Division of Health Education, Office of the Director, at (404) 639-6204.

Answers to Pretest and Challenge Questions

Pretest questions are on page 1. Challenge questions begin on page 3.

Pretest

The Pretest questions (a) and (b) are answered in Challenge answers 1, 2, 3, and 6 below.

Challenge

(1a) The man in Case No. 1 has most likely developed an irritant contact dermatitis from the insulating material (e.g., fiberglass, rock wool). This possibility could be investigated by placing skin scrapings on a microscope slide with 1 to 3 drops of 10% potassium hydroxide (KOH) and examining the specimen under a light microscope using low power. The presence of fibrous strands would confirm the diagnosis. Dermatitis elicited by fibrous glass is variable and depends on individual characteristics and extent of exposure.

(1b) The woman probably has either irritant or allergic contact dermatitis. The basic histopathologic appearance of these two conditions is essentially the same, and differentiating between them by appearance or routine skin biopsy is difficult. However, most paint-stripping products contain one or more of the following compounds: isopropyl alcohol, cresylic acid, methylene chloride, glacial acetic acid, aliphatic hydrocarbons, and aqueous ammonia, all of which tend to be irritants rather than allergens. The presence of mild rather than severe itching, more erythema than vesiculation, localized lesions, and insidious rather than explosive onset are more consistent with irritant contact dermatitis than with allergic contact dermatitis.

(1c) Both the man and the woman should be advised to avoid exposure to the offending substances, at least temporarily. Applying and rapidly removing adhesive or Scotch tape from the man's affected skin may remove the fibers and help relieve the itching.

The contact dermatitis of both patients may be treated using Domeboro's solution (1:40 dilution) or Burow's solution. Dressings soaked with one of these solutions should be applied topically for 15 to 20 minutes, 6 times daily. Topical corticosteroids may be applied, starting with a steroid of low potency and progressing to more potent corticosteroids as needed. Mild sedatives and antihistamines may be administered to relieve itching. Topical or systemic antibiotic therapy may be used to combat secondary bacterial infection. Repeated exposure to UV radiation may be therapeutic in some cases, causing hardening or increased resistance to further irritation.

(2a) The rash of the patient in Case No. 2 is not likely to be due to airborne allergens or irritants in the new office location. Although some cases of allergic or irritant contact dermatitis can develop from exposure to airborne allergens or irritants, the patient's occupational history and the location of the rash do not suggest this etiology. Eyelids, cheeks, nasal folds, and the neck most probably would be involved if an airborne agent in the workplace were responsible. The hand and wrist location suggests contact with an allergen that is handled.

(2b) Yes, the patient's woodworking hobby, with recent introduction of various exotic woods and Japanese varnish (possibly derived from the Japanese lacquer tree), suggests a cross-sensitivity reaction to agents related to *Rhus* plants, to which he is known to be sensitized.

(2c) The patient probably has allergic contact dermatitis. Therapy would be identical to the regimen for irritant contact dermatitis described in (1c) above. Attempts to desensitize sensitive persons have been unsuccessful in most cases.

(3a) Given the rural location and outdoor activities in which the children in Case No. 3 were involved, airborne allergic contact dermatitis to *Rhus*-type plant oleoresins (e.g., poison ivy, poison oak, poison sumac) or pollen could be the cause. However, vesiculation would be expected with allergic contact dermatitis. Patch testing could rule out this diagnosis.

Puncturing lime skins while making sachets during craft class could have exposed the children to psoralens, which are known photoirritants. Immediately after the craft class, the children engaged in outdoor sports. This combination of activities could lead to photosensitivity dermatitis. Because the counselors were involved in a staff meeting during the craft class and did not puncture the limes, only the children were affected.

(3b) One of the most important components in therapy for photoreactions is identification and avoidance of the photoactive agent. When exposure to the offending agent cannot be avoided, sunlight exposure should be minimized. Light exposure can be reduced by wearing protective clothing such as broad-brimmed hats, long sleeves, and tightly woven fabrics, or by using sunblocking agents. Symptomatic topical treatments may also be used.

(4a) The rash of the patient in Case No. 4 is more consistent with chloracne than with acne vulgaris. Acne vulgaris has a different appearance, and its distribution is typically the central face, back, and chest; it seldom affects the buttocks. The sebaceous glands are usually active in acne vulgaris, but chloracne gives the skin a "dry" appearance. Comedones are small in size and number in cases of acne vulgaris, whereas typical straw-colored cysts are almost pathognomonic for chloracne.

The patient's occupation is a potentially relevant factor. A telephone call to a manager at the utility company reveals that old heat exchanger fluids contain PCBs, and in the process of replacing these fluids with less hazardous materials, the workers could have accidental contact with the material. The finding that PCBs are the most probable cause of the patient's chloracne should prompt a health hazard evaluation by the appropriate regulatory authorities and should encourage action to prevent further exposure.

(4b) The chloracnegenic agent should be identified and exposure stopped. Chloracne is resistant to treatment in many cases. Medications used for acne vulgaris are ineffective for chloracne, but oral and topical antibiotics, acne surgery, injection of inflammed cysts with triamcinolone, and dermabrasion of scars may be efficacious. Topical application of retinoic acid (Vitamin A) or 13-cis-isoretinoic acid (Accutane) has been used on carefully selected patients with some success. In addition to treating the skin lesions, examination and testing should be performed to rule out hepatotoxicity, porphyria cutanea tarda, and peripheral neuropathy—all possible systemic effects of PCB exposure. (For further information, see *Case Studies in Environmental Medicine: Polychlorinated Biphenyl [PCB] Toxicity.)*

(5a) Yes, the manufacturing plant could be associated with the children's skin lesions in Case No. 5. A similar outbreak among workers at a manufacturing facility and children in a neighboring school was reported in 1985; a powdered thiadiazole was responsible in that case. Because an etiologic agent for the pigment changes in the children has not been found in routine testing of the water and food at the school, it would be advisable to consider other common sources in the neighborhood, such as the school playground. The nearby chemical manufacturing facility should also be investigated as a possible source, especially because the parents of the two children with more severe manifestations are employed at this plant. The parents could be carrying contamination home on their skin, clothing, and shoes. In addition, the children may be playing in an area with contaminated soil.

(5b) You could begin your investigation by contacting the nurse or health and safety manager at the parent's workplace to determine whether a workplace agent or process is associated with the rashes of some workers. You could request from the manufacturer Material Safety Data Sheets (MSDSs) or other information about the raw materials, byproducts, chemical intermediates, and finished products used or produced at the plant.

The Toxic Chemical Release Inventory (TRI), which is maintained by the U.S. Environmental Protection Agency (EPA) and is available to the public either online through the National Library of Medicine or on CD-ROM, could be used to determine the normal releases from the plant. Plant management, the local EPA, or the local fire department could be consulted to determine whether any accidental chemical releases have occurred recently at this facility. If soil contamination is suspected, soil samples from nearby playgrounds, school yards, or other play areas should be tested. Local or state health officials may be contacted for assistance.

(5c) If the lesions are persistent, large, and cosmetically displeasing, you could refer the children to a dermatologist for consideration of PUVA treatment. Sunscreens and protective clothing can protect areas with depigmented skin and prevent hypopigmentation from worsening.

(6a) The constellation of complaints of the patient in Case No. 6 is consistent with contact urticaria syndrome. Balsam of Peru and various alcohols (especially propyl alcohol and ethyl alcohol) in numerous consumer cosmetic products and benzocaine in many over-the-counter topical analgesic preparations could be causative agents.

(6b) Evaluation might include correlating the history of the illness with probable exposures, serologic studies of circulating IgE, and patch or scratch testing (performed by, or in consultation with, a dermatologist in a setting with resuscitation equipment in case of anaphylactoid reaction).

(6c) Usual treatment for contact urticaria includes advice to avoid suspected or known causative substances and administration of antihistamines. In certain patients, nonsteroidal anti-inflammatory medications have shown some efficacy.

(7a) The constellation of complaints of the couple in Case No. 7 suggests chronic arsenic poisoning. Arsenic toxicity from criminal activity, intentional surreptitious self-injury, occupational exposure, and environmental exposure should be investigated.

On questioning, the couple reveals that they have obtained drinking water from a private well for the past 40 years and that they heat their home with a wood stove fueled with scrap wood. Analysis of the well water reveals arsenic at 0.62 milligrams per liter (mg/L), a concentration significantly above the EPA maximum contaminant level (MCL) of 0.05 mg/L. Ashes collected from the wood stove and soot from the chimney also contain arsenic in concentrations of several hundred parts per million; the most likely source of this contamination is arsenic-containing preservatives in the scrap wood.

(7b) Initial action should be taken to terminate further arsenic exposure; it will be futile to treat the skin lesions (or provide chelation therapy to reduce body burden) if exposure continues. An alternative source of drinking water should be substituted immediately, contaminated lumber should not be burned, and the home should be decontaminated. Advice on abatement and remediation and aid in investigating any other possible sources of arsenic may be obtained from the state or local health department. (For further information on arsenic and arsenic poisoning, see *Case Studies in Environmental Medicine: Arsenic Toxicity.)*

Treatment of the man's basal cell carcinoma may involve radiation therapy or excisional biopsy, including a suitable margin of normal-appearing skin. All tissue removed should be submitted for histologic confirmation of diagnosis and to be certain the tissue borders are free of cancerous cells. The patient should be counseled to avoid prolonged exposure to sunlight and to use sunscreens or protective clothing whenever exposure to sunlight is anticipated.

Glossary*

anaphylaxis. Commonly used to denote the immediate, transient kind of immunologic (allergic) reaction characterized by contraction of smooth muscle and dilation of capillaries due to release of pharmacologically active substances (histamine, bradykinin, serotonin, and slow-reacting substances), classically initiated by the combination of antigen (allergen) with mast cell-fixed, cytophilic antibody (chiefly IgE).

acne mechanica. Acne caused or exacerbated by friction.

acne medicamentosa. Acne caused or exacerbated by several classes of drugs including antiepileptics, halogens, and steroids.

acne venenata. Acne produced by external irritants or drugs internally administered.

acne vulgaris. Simple acne, probably caused by hormonal fluctuations.

bullae (singular bulla). Large bubble-like structures (vesicles) appearing as a circumscribed area of separation of the epidermis from the subepidermal structure, typically filled with serum.

chloracne. Acne-like eruptions due to prolonged contact with certain chlorinated aromatic hydrocarbon compounds.

chloracnegenic agents. Substances that cause chloracne.

comedones. A plug of sebaceous matter, capped with a blackened mass of epithelial debris, filling the pilosebaceous orifice.

dermatitis. Inflammation of the skin.

atopic d. Characterized by the distinctive phenomena of atopy, a Type I allergic reaction, specifically one with strong familial tendencies, caused by allergens such as pollens, foods, dander, and insect venoms, and associated with the Prausnitz-Küstner (IgE class) antibody.

allergic contact d. A delayed type of induced sensitivity (allergy) of the skin with varying degrees of erythema, edema, and vesiculation, resulting from cutaneous contact with a specific allergen.

irritant contact d. Irritation of skin caused by contact with substances that are toxic to epidermal or connective tissue cells; lesions are usually erythematous and papular, but can be purulent or necrotic, depending on the nature of the toxic material applied.

dermatomyositis. A progressive syndrome characterized by muscular weakness with a purplish erythematous skin rash on the face.

eczema. Generic term for acute or chronic inflammatory conditions of the skin, typically erythematous, edematous, papular, vesicular, and crusting; often followed by lichenification and scaling and occasionally by duskiness of the erythema; often accompanied by sensations of itching and burning.

erythema. Inflammatory redness of the skin.

*Adapted from *Stedman's Medical Dictionary*, 25th edition, Baltimore: Williams and Wilkins, 1990. Modified with permission from Williams and Wilkins.

erythema multiforme. An acute eruption of macules, papules, or subdermal vesicles presenting a multiform appearance, the characteristic lesion is typically over the dorsal aspect of the hands and forearms; its origin may be allergic, seasonal, or from drug sensitivity, and the eruption may be recurrent or may run a severe course (Stevens-Johnson syndrome), possibly ending in death.

excipient. An inert substance such as gum arabic, syrup, lanolin, or starch, that acts as a diluent or forms a vehicle for drug delivery.

folliculitis. An inflammatory reaction in hair follicles; the lesions may be papules or pustules.

exfoliative dermatitis. General scaling of the skin, usually with erythema.

hapten. Incomplete or partial antigen; an antigen that is incapable, alone, of causing the production of antibodies.

hives. See urticaria.

hyperkeratosis. Hyperkeratinization; hypertrophy of the horny layer of the epidermis.

hyperpigmentation. Increased pigmentation of the skin.

hypertrichosis. Growth of hair in excess of normal.

hypopigmentation. Decreased pigmentation of the skin.

keratosis. Any lesion on the epidermis marked by the presence of circumscribed overgrowths of the horny layer.

lichenification. Leathery induration and thickening of the skin with hyperkeratosis, due to a chronic inflammation caused by scratching or long-continued irritation.

leukoderma. An absence of pigment, partial or total, in the skin.

macular. Relating to or marked by a small, discolored patch or spot on the skin, neither elevated nor depressed below the skin's surface.

malar crescent. Around the cheek or cheekbones.

melanin. Pigment that occurs in the hair, skin, or retinas.

melanocytes. Pigment cells of the skin.

melanoderma. An abnormal darkening of the skin by deposition of excess melanin, or of metallic substances such as silver and iron.

melanoma. A malignant neoplasm derived from cells that are capable of forming melanin, which may occur in the skin of any part of the body; in the early phases, the lesion is characterized by proliferation of cells at the dermal-epidermal junction, and the neoplastic cells soon invade adjacent tissue extensively. Melanomas frequently metastasize widely; most examples of this neoplasm occur in adults and may originate de novo or from a pigmented nevus or malignant lentigo.

milia (singular *milium*). Sebaceous tubercle; whitehead; a small subepidermal keratin cyst, usually multiple, therefore commonly referred to in the plural.

miliaria. An eruption of minute vesicles and papules due to retention of fluid at the mouths of the sweat follicles.

nummular. Marked by circular or oval lesions.

papules. Small, solid elevations on the skin.

photoallergy. Sensitization of the skin to light.

phototoxicity. The condition arising from overexposure to ultraviolet light.

pruritis. Itching.

psoralens. Furo[3, 2-g]coumarin; a phototoxic chemical derived from fruits of the citrus family (e.g., limes).

psoriasis. A condition characterized by the eruption of circumscribed, discrete and confluent, reddish, silvery scaled macropapules.

Rhus. A genus of trees and shrubs (family Anacardiaceae) containing various species that are used for their ornamental foliage; poison ivy, poison oak, and poison sumac belong to this genus.

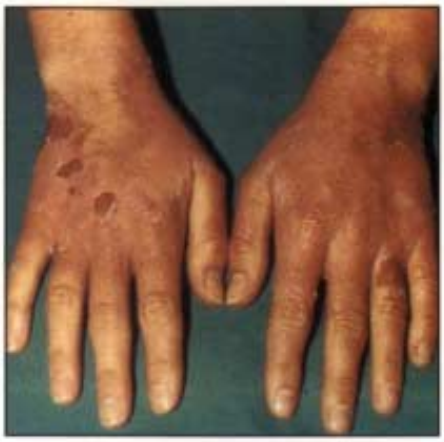
solar elastosis. Degenerative change in elastic tissue of the dermis due to repeated or constant exposure to sunlight over a period of years.

urticaria. Hives; an immediate eruption of itching wheals, which may be due to physical and chemical agents, foods or drugs, foci of infection, or psychic stimuli.

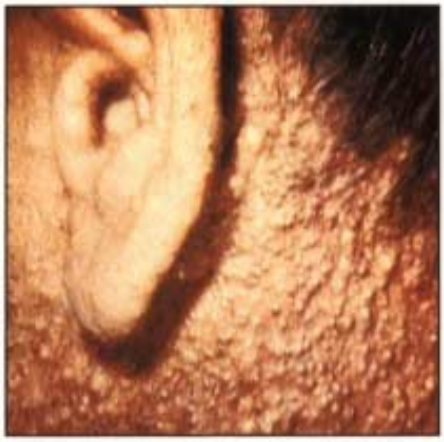
urticaria syndrome. Consists of the typical urticarial rash with systemic involvement.

vesiculation. Blistering.

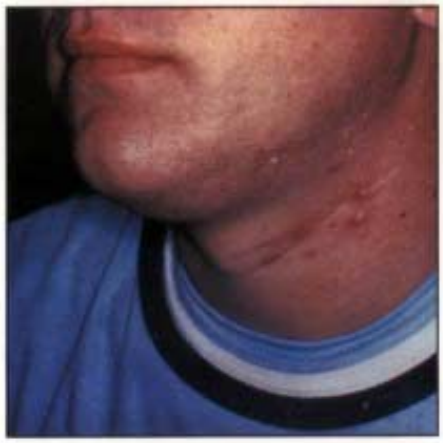
vitiligo. The appearance on the otherwise normal skin of loss of melanin pigment with white patches of varied sizes, often symmetrically distributed; the skin bordering the affected sites is usually hyperpigmented, and hair in the affected areas is usually white.



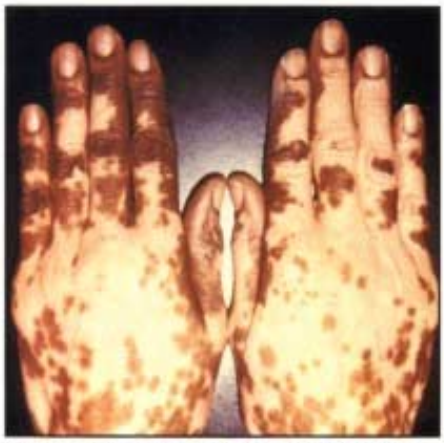
7. **Photosensitivity contact dermatitis**—Many chemicals need light to activate and produce the complete phototoxin or photoallergen. Psoralens in limes produced this vesicular phototoxic dermatitis in a bartender who squeezed limes all afternoon in direct sun.



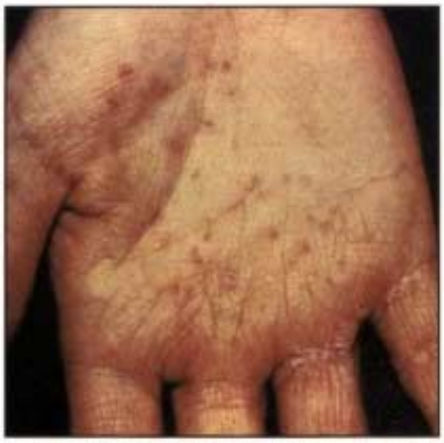
8. **Chloracne**—Chloracne is a refractory type of acne caused by certain halogenated aromatic chemicals; it can be accompanied by systemic toxicity. Chloracne in this herbicide production worker involved almost every follicular orifice on his face and neck, producing comedones, papules, and cystlike lesions.



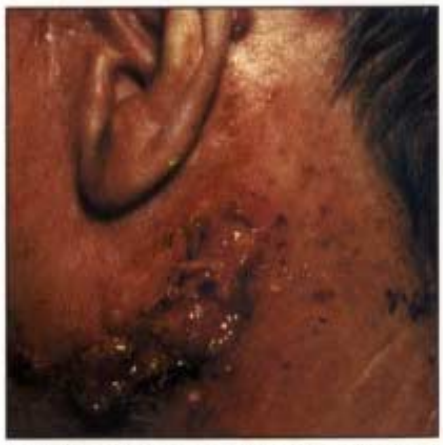
9. **Acne vulgaris**—Acne-prone workers who have contact with oils frequently have poral occlusion problems. Hot, humid environments may cause sufficient hydration and swelling to predispose the skin to acne. This worker's head was positioned in such a way as to constantly crease one side of his neck, causing recurrent deep lesions in that one location.



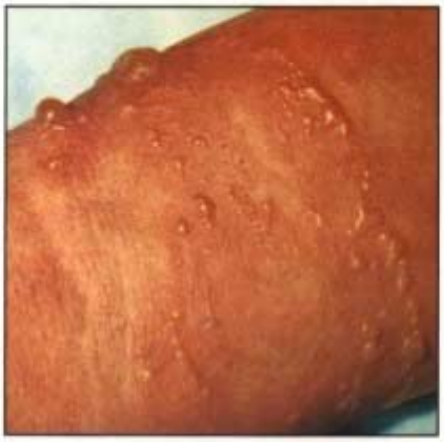
10. **Hypopigmentation**—The hands of this hospital maintenance worker are depigmented from contact with a phenolic germicidal detergent. Irritation or sensitization to the chemical is not a prerequisite for pigment loss. The loss of pigment may be permanent.



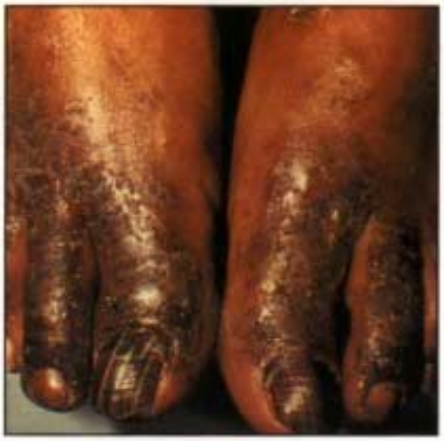
11. **Contact urticaria**—Contact urticaria is an unusual urticarial or wheal-and-flare response occurring upon external contact with certain agents. This is the clinical presentation of a worker sensitive to ground meat. The reaction developed within 30 minutes. Some cases of contact urticaria involve immunologic mechanisms; others do not. Itching may be the only manifestation.



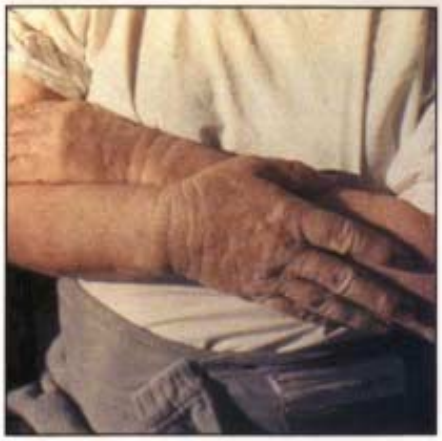
12. **Skin neoplasms**—Skin cancer is the most common form of cancer. Skin tumors, such as this ulcerating squamous cell carcinoma, most frequently arise after years of occupational exposure, or they are common complications from long-term exposure to solar radiation.



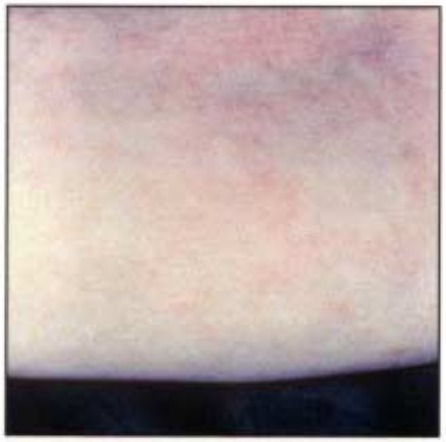
1. **Acute irritant contact dermatitis**—Exposure to a strong irritant, ethylene oxide, produced this markedly swollen arm and an acute vesiculo-bullous dermatitis. A similar pattern may be seen with contact allergy.



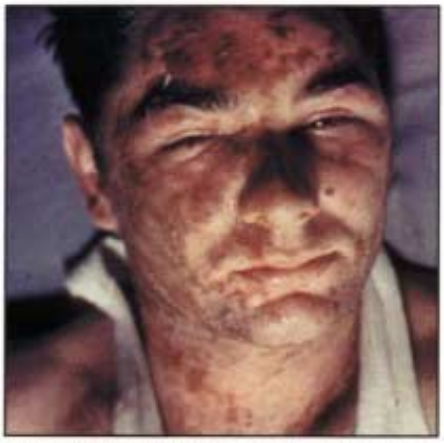
2. **Subacute irritant contact dermatitis**—This patient developed a bilateral and symmetric subacute dermatitis from the rubber accelerator, mercaptobenzothiazole, which was leached from the rubber portion of his work shoe due to sweating. Some edema and erythema with an eczematous eruption can be noted.



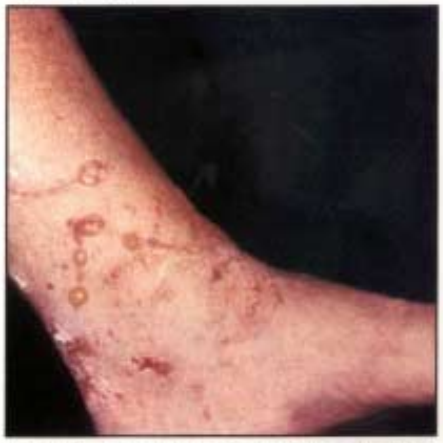
3. **Chronic irritant contact dermatitis**—The hands, wrists, and forearms are the most frequent sites of involvement in cases of industrial contact dermatitis. The hands and wrists of this worker show the effect of long-term exposure to a solvent, kerosene, which he used to clean his skin. The skin is markedly thickened, hyperpigmented, dry, and fissured. Itching is usually a major symptom.



4. **Fibrous glass contact dermatitis**—Contact with fibrous glass, particularly large-fiber diameter, can produce itching; lesions may not be visible except for secondary effects from scratching or rubbing. In some persons, small erythematous papules may occur where the spicules have penetrated, as shown here.



5. **Allergic contact dermatitis**—This severe allergic contact dermatitis was due to a phenol-formaldehyde resin. These resins are used as bonding agents for foundry sand, in electrical devices, and in molded and cast plastic articles. The resins also can produce irritant reactions.



6. **Poison ivy contact dermatitis**—The *Rhus* genus of plants includes poison ivy, poison oak, and poison sumac. Poison ivy dermatitis may be acquired from direct contact with the plant or from the smoke of burning poison ivy plants.